REPORT ON PATHOLOGY AND PATHOLOGICAL ANATOMY.

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Pathology.

"Waxy Degeneration" of Muscular Fibre.—Dr. Weihl (*Virchow's Archiv*, 1874, p. 253) gives the results of a series of observations concerning this condition, first mentioned by Bowman in 1841.

The term was applied by Zenker, in 1864, to that condition of striated muscular fibre where the contents of the primitive fibril are converted into homogeneous masses of varying form and size, possessing a dull, waxy lustre. These masses are quite brittle, and do not essentially differ, chemically, from the contents of normal muscular fibre. He regarded this change as a nutritive disturbance, produced by fever, and due to the rapid reception of new material by the contractile substance. It was observed in typhoid and scarlet fevers, acute miliary tuberculosis, cerebro-spinal meningitis, articular rheumatism, tetanus, &c.; also in the arms of an insane person who had been confined in the straight-jacket. Other observations, before and since, show that this condition has been seen in almost all febrile diseases, in cases of injury, and in the vicinity of morbid growths.

Its origin has been regarded by some as purely mechanical; others considered that the process consisted in a coagulation of the myosine, with subsequent contraction. The change has also been viewed as merely a *post-mortem* one. Cohnheim, however, found it in the tongue of the live frog twenty-four hours after the local supply of blood had been cut off, and quite independent of any direct mechanical violence.

In Weihl's experiments, the frog was used, and it was ascertained that changes resembling, and probably identical with, those of "waxy degeneration" could be produced in the tongue of the live frog in various ways. Since they could be produced voluntarily and immediately, it seemed evident to him that they could not be regarded as a degeneration or as an inflammation. He further considered them as probably due to a coagulation of the contractile substance of the muscle.

In this connection, the investigations of Popoff (*Centralblatt*, 1873, No. 44) are interesting. He observed the effect of polarized light on muscular fibre. The double refracting substance of the muscular fibre was not altered by the "waxy degeneration." He concludes that this change in infectious diseases is rather an appearance accompanying other signs of inflammation of the muscular fibre than an actual process of degeneration.

Fatty Degeneration of the Heart caused by Anæmia.—It is now long since Virchow called attention to the anatomical peculiarities of that form of anæmia to which the term chlorosis is applied, and within a recent period he has again referred to this matter. He regards it as very probable that regularly in such cases there exists, congenitally, a lack of development of the vascular apparatus, especially of the heart and aorta; so that, at the outset, these are abnormally small, and are subject to pathological changes, fatty degeneration, &c., at a very early period.

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Cases have been recorded where an extreme degree of anæmia has arisen and terminated fatally, the only evident lesion being a fatty degeneration of the heart. Gusserow was among the first to call attention to this condition, and reported *(Archiv für Gynäkologie*, 1871, p. 218) five cases where an extreme degree of anæmia developed during pregnancy, apparently without cause, and terminated fatally. In three cases, the heart was found to be fatty degenerated. Biermer soon after called attention to this "progressive pernicious anæmia," and distinctly separated it from simple anæmia. He spoke of its clinical aspects, the pallid skin, the œdematous feet, hands and face, the debility, dizziness and palpitation. There was loss of appetite and occasional diarrhœa. Attacks of fever were noticed, though of no tpyical character. An anæmic souffle was heard, often so loud as to suggest valvular disease. Capillary hæmorrhages occurred in the retina and skin, more rarely in the kidneys. Minute cerebral hæmorrhages were suggested by slight and temporary paralytic attacks. Dropsical effusions occurred towards the close, and intervals of delirium.

Notwithstanding the excessive anæmia, there was no diminution in the amount of fat. The only anatomical changes were the fatty degeneration of the heart and the hæmorrhages. He considered the frequent presence of intestinal catarrh a cause of the anæmia.

Ponfick *(Berliner Klinische Wochenschrift*, 1873, No. 1) regarded this subject from an anatomical point of view, and called attention to a peculiar form of spotted fatty heart, of normal dimensions and with healthy valves, which occurred more particularly in females, from the twentieth to the fortieth year, and which was associated with a marked degree of anæmia. To this, he applied the term anæmic fatty heart. The alterations of the bloodvessels found by Virchow in chlorosis were at times present, but to a slight degree. Changes in the other organs of the body were of relatively trivial importance and were apparently secondary. The gland cells of the liver, kidneys and stomach presented a greater or less degree of fatty degeneration. Jaundice was frequently present. The total amount of blood was apparently diminished, the red corpuscles decidedly so ; likewise the fibrine. Evidence of dropsy was almost constant, as hydrothorax, anasarca or ascites. Ponfick found that these conditions occurred in women with protracted convalescence after delivery, in cases of acute disease, in chronic gastric or intestinal affections with exhausting diarrhœa, and, finally, in persons who had suffered from the loss of blood.

Immermann *(Deutsche Archiv für Klinische Medicin*, 1874, p. 209) regards the disease as different from other forms of anæmia, owing to the lack of a sufficient cause ; its excessive degree and union with alterations of the organs of circulation ; the occurrence of fever without an anatomical basis ; its progressive character and fatal result. He considered that the absence of emaciation was of great value in the differential diagnosis. Ponfick, in several instances, from the extreme pallor of the corpse, or of an organ, was able to anticipate the fatty condition of the heart. Immermann considers the disease to be allied rather to chlorosis and leucæmia, though in no way to be identified with either. It lacks the splenic and glandular swelling of the latter, and is distinct from Addison's disease in that there is no discoloration of the skin. From acute albuminuria, it differs in that there is little or no albumen in the urine.

Its etiology is exceedingly obscure. Immermann calls attention to the occurrence of the greater number of cases hitherto reported in a limited district, the canton of Zurich, and lays weight upon this fact, though unable to recognize an essential or specific cause. Other cases have occurred elsewhere, however, at Dresden and Heilbronn, and the etiological value of a limited local disposition must be regarded as slight.

Perl (*Virchow's Archiv*, 1874, p. 39) endeavored to prove, experimentally, that anæmia resulting from loss of blood would give rise to this form of fatty heart. Dogs were used, a certain number being bled a few times, but to a large extent each time. From the others, small amounts of blood were drawn frequently. The animals of the first series gradually fell into a state of marasmus, lost their appetite, became debilitated and finally died from exhaustion. In some instances, œdema occurred; there was no evidence of fever. The heart showed generally a fatty degeneration of the muscular fibres, most marked in the papillary muscles. The dogs, from whom small quantities of blood were frequently drawn, recovered, were killed, but their hearts were apparently unaltered.

Pathological Anatomy.

Fatty Emboli of the Lungs.—That embolism from oil globules might occur in the pulmonary as well as in the general circulation, has long been recognized. Experiments have shown that the intra-venous injection of large quantities of oil would produce death within a few minutes, while that of smaller quantities would give rise to death within a few hours, apparently due to œdema of the lungs. The case of Wagner and the subsequent experiments of Busch have directed a closer attention to this subject, and have been of great influence in suggesting a rational theory for the cause of death in a certain number of those cases of severe injury where such has been attributed simply to "shock."

Wagner's case was that of a man whose thigh was broken. Death afterwards occurred, preceded by coma, and many of the capillaries in different parts of the body were found to be plugged with oil globules. Investigating the matter experimentally, Busch found that, after breaking bones and crushing the marrow, fat drops were set free and were apparently taken up by the veins. They were then carried to the lungs and obstructed the pulmonary capillaries, producing death by œdema of these organs.

Bergmann (*Berliner Klinische Wochenschrift*, 1873, No. 33) adds another case to the series. His patient fell from a height of thirty feet; there resulted a comminuted fracture of the thigh. After a short time, pain in the chest was complained of; later, a frothy, bloody expectoration was ejected, the respiration increased in frequency, the lips became livid, the temperature rose, and fine moist râles were heard throughout both chests. Bergmann made a diagnosis of acute œdema of the lungs, caused by fatty emboli, eliminating traumatic pneumonia and pulmonary hæmorrhage. Death occurred seventy-nine hours after the accident. At the autopsy, both lungs were found to be hyperæmic, œdematous, dotted with small dark spots, and containing hæmorrhagic infarcts of the size of a pin's head.

The microscope showed the arteries and capillaries to be filled to an

extreme degree with liquid fat. The broken bones were very much comminuted, the marrow crushed, and free fat, mixed with blood, was found at the seat of fracture.

Fatty embolism may occur in other ways, as is shown by a communication from Egli (*Untersuchungen aus dem pathologischen Institut zu Zürich. Jahresbericht der Gesammten Medecin*, 1873, p. 214). He reports two cases where fat was found in the pulmonary vessels in the form of drops and cylinders. In one case the condition was extreme, and he states that, during the last four or five days previous to death, the patient suffered from violent dyspnœa. The source of fat in these two cases was considered to be thrombi in the right heart, which had undergone puriform softening. The degenerated clots were found to contain abundant fat drops, large and small. He suggests that the absence of fatty emboli in the general circulation might be due to the action of the heart being enfeebled to such a degree as to be unable to force the fat drops through the pulmonary capillaries.

Croup and Diphtheria.—As the term diphtheria, or diphtheritis, has become rather vague in the course of time, Senator (*Sammlung Klinischer Vorträge*, No. 78, 1874) would use the term "cynanche contagiosa" to designate an "acute, contagious disease, which occurs chiefly among children, usually beginning with fever, and producing certain alterations of the mucous membrane at the junction of the respiratory and digestive tracts, or in those parts of both tracts in the immediate vicinity of this place of union; that is, alterations of the mucous membrane of the tonsils, uvula and soft palate, base of the tongue, larynx and bronchi, the posterior wall of the pharynx or of the nasal cavity."

The local anatomical changes present numerous variations, dependent upon the quantity and condition of the contagium, possibly upon its stage of development, and upon the disposition of the patient or of the organ affected. A simple catarrh of the mucous membrane is the mildest expression; this may terminate in complete recovery within a few days. That this is no ordinary catarrh is to be inferred from its epidemic occurrence, often in the same house with the severer forms of cynanche. Further, the glands of the neck are usually swollen, and, lastly, it may be associated with the development of the severer forms.

There are, first, small round or longitudinal grayish-white, membrane-like patches seated upon the catarrhal portions of the mucous membrane, and which can readily be removed, thus disclosing an apparently unaltered mucous membrane. These patches rarely remain longer than a day, as convalescence or the more serious condition then follows. They are composed of tesselated epithelium, more or less altered, with which are found vegetable spores. It is of great importance that there is no evidence of an inflammatory process in these patches.

Finally, the remaining form also presents flat, grayish-white patches in the pharynx. These are more intimately connected with the mucous membrane beneath, and, when separated, are found to be merely the upper surface of a shallow ulcer, which extends superficially. Pain and slight bleeding are produced by attempts at their removal. The tissues beneath are infiltrated with cells, contain extravasated blood, while the superficial portions, the patches, represent dead bits

of tissue, which are more or less loosely connected with the parts beneath. These patches are composed of epithelial cells of varying size and in various stages of destruction, of pus and blood corpuscles, of blood coloring matter; also of granular detritus and spores. They are rather to be compared with the gangrenous masses from a bed-sore, and the process giving rise to them is best called an acute gangrene or diphtheritic inflammation.

The transition between this stage and the preceding is a gradual one and may be overlooked; at the same time, it is not essential that the epithelial patches should have preceded, more particularly where the disease is violent and progresses more rapidly.

Putrefaction readily takes place at the seat of the inflammation, and may advance to such a degree as to give a most marked instance of moist or putrid gangrene. This is to be considered merely as a higher degree of the diphtheritic form.

In the larynx, trachea and larger bronchi, the epidemic of cynanche likewise produces a catarrhal and diphtheritic inflammation. The latter occurs almost exclusively in the upper part of the larynx, as far down as the true vocal cords, and on the epiglottis. The anatomical structure of these parts, as well as the rapidly fatal result in general, prevent such deep destruction as is found in the tonsils. The epithelial false membrane is not found in the air passages, owing to the delicate nature of the cells there, especially of the ciliated epithelium, which is never detached in continuous masses.

In the air-passages, however, the fibrinous or croupous false membrane is found, which seldom if ever occurs in the pharynx. This is essentially composed of a coagulated albuminate, so-called fibrine, pus corpuscles, and an occasional red blood corpuscle. The absence of epithelium and spores is notable. If this croupous membrane is fresh, scarcely any spores are found. The mucous membrane beneath it is apparently unaltered. The epithelial layer may often be recognized, though at times it is wanting. The deeper layers of the mucous membrane are infiltrated with pus corpuscles, but there is no extravasated blood.

The anatomical alterations produced by cynanche contagiosa are these: catarrh, epithelial shreds only in the pharynx, diphtheritic patches from diphtheritic inflammation above the vocal cords, and fibrinous false membranes from croupous inflammation in the respiratory tract below the vocal cords.

During the epidemic of cynanche, and dependent upon it, there has been observed a diphtheritic inflammation of other mucous membranes —of the conjunctiva, for instance, or of the genital mucous membrane. It is not, therefore, to be concluded that every diphtheritic inflammation of these membranes, even when endemic, is due to the cynanche. A diphtheritic inflammation may occur upon the surface of any wound or ulcer, under certain local or general conditions. In cynanche, it is the rule for these different forms of inflammation to occur side by side and to run into each other. When the child dies with cynanche, the *post-mortem* examination usually discloses membranous patches (diphtheritis) in the upper part of the larynx, the fibrinous exudation (croup) lower down in the air passages, and, still further downwards, a catarrh. It is usually impossible to say where the croupous membrane ceases and the diphtheritic membrane begins; nor is it to be

stated that the former represents a less, the latter a more severe result of the cause of the disease. The process usually begins on the tonsils, soft palate or posterior wall of the pharynx, and is very often limited to these parts. It is very rare for the larynx to become affected without a previous inflammation, or a very rapid subsequent affection of the pharynx. The inflammation may extend from the pharynx into the nose or larynx, but rarely into the œsophagus. It may extend from the nasal cavity to the conjunctival mucous membrane. Senator does not absolutely reject the view that there may be a croup limited to the air-passages, and distinct from the cynanche or diphtheria. There seems no doubt that chemical irritants may produce a croupous inflammation of the air-passages, and observers, up to a comparatively recent period, even now, state that in certain epidemics, while the air-passages are affected by the croupous inflammation, there is no alteration, whatsoever, of the pharynx. At the same time, he maintains that "in by far the greatest majority of all cases, at present, at least, the croupous inflammation of the larynx and air-passages is the result of the cynanche contagium, and occurs almost exclusively in epidemics of cynanche simultaneous with its other anatomical forms." Hence, in general, croup and diphtheria represent merely forms of one and the same disease, cynanche.

Senator regards it as indisputable that the contagious material of the disease is attached to the mucus, pus and shreds of tissue from the diseased parts of the throat, and as not improbable that the same is living and organized. At the same time, the micrococci found in this disease can in no way be distinguished from the organisms found in any putrefactive process, and their inoculation gives rise to no differing results; hence, they cannot be regarded as possessing any specific nature. The idea that the micrococci of putrefaction cause the cynanche, or that micrococci are the first and only cause of the disease, is opposed by the fact that they are not found in any quantity below the vocal cords. In addition, the disease almost never extends into the œsophagus, a road apparently so direct. Finally, the same anatomical alterations of the pharynx may occur in scarlatina and cynanche, while the alterations of the air-passages are very rare in the former disease.

It is considered quite probable, from reasons advanced, that the peculiar form of diptheritic inflammation is due to micrococci. Whenever diphtheritis occurs, the micrococci are found, almost without exception, and by inoculating an inflamed part with putrid material, containing micrococci, a diphtheritic inflammation may be produced. For the germs to become active, they must be sufficient in quantity, in a state of activity, and with a capacity for development, and the tissue must be capable of receiving them. In the pharynx, all these conditions are present. In the respiratory tract, the ciliary movement prevents the colonization of the micrococci, and the carbonic acid gas possesses antiseptic properties.

According to Senator, the unknown contagium of cynanche disposes to a violent inflammation of the parts affected, as that of scarlatina does to the pharyngeal mucous membrane, or that of measles to the mucous membrane of the larger air-passages, nose and eyes. As a

result of the inflammation, the epithelium is loosened and elevated, and losses of substance occur. The spores, always abounding in the pharynx, enter the tissues, and give rise to the death of the inflamed, superficial layers, and the diphtheritis occurs. The latter is, therefore, secondary, and may follow other forms of pharyngeal affections, as in scarlatina, variola, syphilitic ulcers, &c. The diphtheritis once established, and the micrococci favored with so suitable a soil, the air may become readily infected by them, and every ulcer exposed to this air may readily become diphtheritic. There is no necessity, however, that the cynanche poison should be therewith transmitted.

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